

Brief Communications

Late iatrogenic coronary stenosis after selective intraoperative antegrade myocardial protection for stentless aortic valve replacement

Simon Maltais, MD, MSc,^{a,b} Ismail El-Hamamsy, MD,^{a,b} Anique Ducharme, MD,^{a,c} Michel Carrier, MD,^{a,b} Michel Pellerin, MD,^{a,b} and Louis P. Perrault, MD, PhD,^{a,b}
Montreal, Quebec, Canada



Dr Maltais

Local injuries to the arterial wall induce periadventitial angiogenesis stimulating intimal thickening.¹ Injuries of the arterial wall disrupt normal vascular hemostasis, promoting intimal hyperplasia.²

We report the case of a 59-year-old man with concomitant severe stenosis of the proximal iatrogenic left main trunk and right coronary artery ostium from selective intraoperative antegrade myocardial protection 10 years after aortic valve replacement (AVR) for aortic stenosis.

Clinical Summary

On May 17, 2005, a 59-year-old man was transferred to the Montreal Heart Institute with exertional chest pain that had progressed over 6 months. The patient had already been operated on in another center in 1995 for severe symptomatic rheumatic aortic stenosis. During the procedure, the aorta had been crossclamped and high-potassium cold blood cardioplegic solution was delivered through the root in a classic antegrade fashion: selective infusion of cardioplegic solution into the left and right sides of the heart via the coronary ostia with a 6-mm balloon-tipped cannula every 15 minutes. Faced with the patient's refusal of anticoagulation therapy, a Freestyle No. 25 bioprosthesis (Medtronic, Mississauga, Ontario) was implanted. The patient had a good evolution and was discharged from the referring hospital with no residual symptoms.

The patient was admitted to our institution and transthoracic echocardiography revealed severe rheumatic mitral stenosis with a calculated valve area of 0.85/cm². The calculated aortic valve area was 1.7 cm² and no insufficiency was noted. A scalloped subcoronary bioprosthesis was in good position (Figure 1) with a trivial peak gradient of 9 mm Hg. The patient's first coronary arteriogram showed proximal severe filiform infiltration of the left main trunk and stenosis of the right coronary ostium (Figure 2, A and B). Ventricular function was preserved and the mitral valve was not suitable for repair.

On May 18, 2005, the patient had successful mechanical mitral valve replacement with complete myocardial revascularization. A median sternotomy was performed, and the left internal thoracic artery was mobilized for bypass. The internal saphenous vein was harvested by endoscopy. Double vein cannulation was performed and an antegrade infusion of high-potassium blood cardioplegic solution was given simultaneously through the coronary sinus and venous bypasses for cardiac arrest. Venous distal anastomosis on the right coronary and first marginal arteries was performed with 7-0 Prolene sutures (Ethicon, Inc, Somerville, NJ). Because of a long, nonaccessible intramyocardial portion of the anterior descending inter-ventricular artery, the internal thoracic artery was anastomosed to the first diagonal artery. Left auriculotomy was performed and the mitral valve correctly visualized. The anterior

From the Research Center,^a Department of Surgery,^b and Department of Cardiology,^c Montreal Heart Institute and Université de Montréal, Montreal, Quebec, Canada.

Received for publication Feb 6, 2006; revisions received March 23, 2006; accepted for publication March 28, 2006.

Address for reprints: Louis P. Perrault, MD, PhD, Research Center, Montreal Heart Institute, 5000 Belanger St, Montreal, Quebec, HIT 1C8, Canada (E-mail: louis.perrault@icm-mhi.org).

J Thorac Cardiovasc Surg 2006;132:420-1
0022-5223/\$32.00

Copyright © 2006 by The American Association for Thoracic Surgery

doi:10.1016/j.jtcvs.2006.03.049

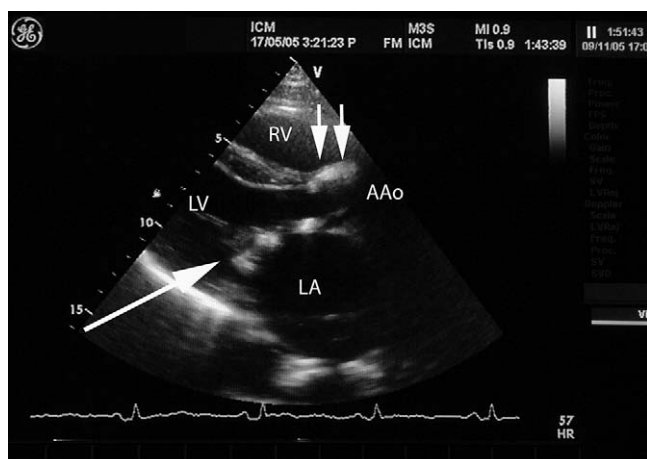


Figure 1. Transthoracic echocardiography showing Freestyle No. 25 bioprosthesis in the aortic position 10 years after implantation (arrowheads). RV, Right ventricle; LV, left ventricle; LA, left atrium; AAo, ascending aorta.

retracted portion of the valve was excised and a St Jude Medical No. 25 mechanical valve (St Jude Medical, Inc, St Paul, Minn) was installed with interrupted 2-0 Ti-Cron sutures (Davis & Geck, Danbury, Conn). The left auriculotomy was closed with 2 continuous 3-0 Prolene sutures. Weaning from extracorporeal circulation was progressively done and the residual 2 proximal anastomoses were completed. Total aortic crossclamping time was 82 minutes and extracorporeal circulation was stopped after 118 minutes with minimal use of inotropic drugs. Postoperative evolution was excellent and the patient returned to the referring center 5 days after surgery for adjustment of anticoagulation therapy.

Conclusion

We report the case of a 59-year-old man with delayed symptomatic severe stenosis of the proximal iatrogenic left main trunk and right coronary artery ostium from selective intraoperative antegrade myocardial protection after AVR for aortic stenosis. The patient underwent successful myocardial revascularization and mitral valve replacement. Our group previously published 7 cases of early left main iatrogenic coronary stenosis after intracoronary administration of cardioplegic solution ranging from 4 to 11 months (mean 7.3 months) after AVR.³ This case reflects an unusually late

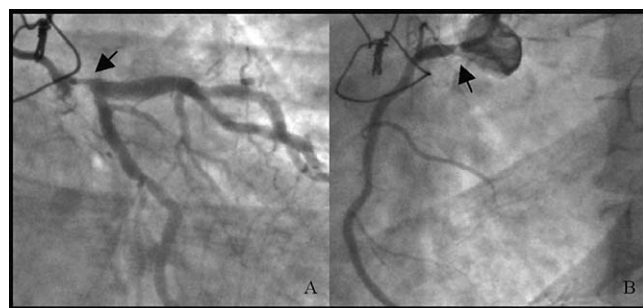


Figure 2. A, Coronary arteriogram showing proximal severe filiform infiltration of the left main trunk (A) and severe stenosis of the right coronary ostium (B).

presentation of mechanical iatrogenic damage to coronary arteries after selective intraoperative antegrade delivery of cardioplegic solution. Animal models have shown that coronary endothelial dysfunction is an early marker of intimal thickening.⁴ Functional undisturbed endothelium is critical for the prevention of vasculopathy.⁵ Surgically induced endothelial injury may occur intraoperatively from mechanical manipulation, ischemia, hypothermia, and exposure to cardioplegic solutions. The use of intracoronary cannulas to deliver cardioplegic solution must always be guided by the concern of inducing as little trauma as possible. This emphasizes that vascular endothelium is a complex modulator of biologic systems and is critical in cardiovascular pathophysiology.

References

1. Khurana R, Zhuang Z, Bhardwaj S, Murakami M, De Muinck E, Yla-Herttuala S, et al. Angiogenesis-dependent and independent phases of intimal hyperplasia. *Circulation*. 2004;110:2283-6.
2. Ganghadharan SP, Eslami MH, Weiss IP, Sui X, Conte MS. Monocyte adhesion to balloon-injured arteries: the influence of endothelial cell seeding. *J Vasc Surg*. 2001;33:1247-54.
3. Chavanon O, Carrier M, Cartier R, Hébert Y, Pellerin M, Perrault LP. Early reoperation for iatrogenic left main stenosis after aortic valve replacement. *Cardiovasc Surg*. 2002;10:256-63.
4. El-Hamamsy I, Stevens LM, Vanhoutte PM, Perrault LP. Injury of the coronary endothelium at implantation increases endothelial dysfunction and intimal hyperplasia after heart transplantation. *J Heart Lung Transplant*. 2005;24:251-8.
5. Zilla P, von Oppel U, Deutsch M. The endothelium: a key to the future. *J Card Surg*. 1993;8:32-60.